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Biologic and epigenetic impact of commuting to work by car or using public transportation: A case–control study

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ABSTRACT

Background and aims. Commuting by public transportation (PT) entails more physical activity and energy expenditure than by cars, but its biologic consequences are unknown.

Methods. In 2009–2010, we randomly sampled New York adults, usually commuting either by car (n = 79) or PT (n = 101). Measures comprised diet and physical activity questionnaires, weight and height, white blood cell (WBC) count, C reactive protein, (CRP) gene-specific methylation (*IL-6*), and global genomic DNA methylation (LINE-1 methylation).

Results. Compared to the 101 PT commuters, the 79 car drivers were about 9 years older, 2 kg/m^2 heavier, more often non-Hispanic whites, and ate more fruits and more meats. The 2005 guidelines for physical activity were met by more car drivers than PT users (78.5% vs. 65.0%). There were no differences in median levels of CRP (car vs. PT: 0.6 vs. 0.5 mg/dl), mean levels of WBC (car vs. PT: 6.7 vs. 6.5 cells/mm^3), LINE-1 methylation (car vs. PT: 78.0% vs. 78.3%), and promoter methylation of *IL-6* (car vs. PT: 56.1% vs. 58.0%).

Conclusions. PT users were younger and lighter than car drivers, but their commute mode did not translate into a lower inflammatory response or a higher DNA methylation, maybe because, overall, car drivers were more physically active.

Introduction

Work or school commute offers a logical option to integrate more physical activity in daily life as a means of counterbalancing the sedentary forces behind the on-going obesity epidemic. Even though biking and walking to work and school would be most effective, for most Americans the choice, if any, is between car and public transportation (PT). PT users walk and climb stairs more than car commuters do, as a result of moving to, from, and within stations (Besser and Dannenberg, 2005; Edwards, 2008; Lachapelle and Frank, 2009; Ogilvie et al., 2004). We have documented the higher physical energy expenditure of PT users during their work commute compared to car drivers (Morabia et al., 2009, 2010).

After the introduction of a new commuter light rail transit in North Carolina, MacDonald et al. (2010) found that the rail commuters had an 18% reduction in body mass index compared to those who kept commuting by car, corresponding to the loss of 6.5 lb for a person 5′5″ (165 cm) tall over 7 months. This was equivalent to an average excess energy expenditure of about 100 kcal/day, compatible with simulation studies suggesting that an average loss of 100 kcal/day can stabilize the progression of a population's weight (Hill et al., 2003; Morabia and Costanza, 2004).

Increased energy expenditure and potentially associated loss of body weight can reduce inflammatory responses, as assessed by total white blood cell (WBC) count and C-Reactive Protein (CRP), (Ford, 2002; Hammett et al., 2004; Kasapis and Thompson, 2005) and epigenetic markers such as global genomic DNA methylation (Zhang et al., 2011a) and gene-specific methylation (Coyle et al., 2007). Inflammatory processes are involved in atherogenesis (Mora et al., 2007) and carcinogenesis (Coussens and Werb, 2002; Rogers et al., 2008). There is, however, no research yet evaluating whether commute-specific physical activity is involved in chronic disease pathways.

This study was therefore designed and conducted to provide first evidence about the inflammatory markers and epigenetic characteristics of random samples of people commuting to a college campus in Queens, NY, either by car or by PT.

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Methods

Participants were identified using a campus-wide survey about commuting habits which had been performed every winter since 2007 (Morabia and Zheng, 2009). Over the years, 4213 respondents agreed to be contacted for research projects related to transportation. They comprised 43% of car commuters and 51% of PT commuters; 6% only commuted by bike, motorcycle, or walked. We recruited and financially remunerated for time a sample of those who were nonsmokers, had no work-related exposure to air pollutants, were students or employees of Queens College, City University of New York, and commuted 5 days/week to and from the campus either by car or by PT. Subjects were not eligible if they had recently used anti-inflammatory drugs, such as aspirin, NSAID, or corticoid drugs.

The car and PT commuters were sent several recruitment emails and were entered into the study in the order in which they volunteered between September 2009 and December 2010. The initial objective was to recruit 100 car ("cases") and 100 PT commuters ("controls").

WBC, CRP, LINE-1 and *IL-6* DNA methylation, diet (including alcohol intake), overall energy expenditure, and body weight were measured on all participants. Body weight and height were measured using a Detecto® medical scale and gauge. The protocol had been approved by the Institutional Review Board of Queens College.

Blood measurements

Blood was obtained by venipuncture at Queens College by a nurse into coded EDTA-tubes. WBC count (cells/mm^3) and hs-CRP (mg/dl) were assayed by a commercial clinical laboratory (Quest). WBC counts were determined immediately after collection, while, for the other measures, a 7 ml tube was taken in a refrigerated box to Columbia University, plasma and WBC isolated and stored at −80 °C. Samples were analyzed in batches at the middle and end of the study. Each batch had a mix of PT and car commuter bloods.

DNA was extracted from the WBC using FlexiGene DNA Kits (Qiagen, Valencia, CA) at Columbia University. Bisulfite modification was conducted using an EZ DNA Methylation-Gold kit (Zymo Research, Irvine, CA) following the manufacturer's recommendations. The biotinylated PCR products were purified and pyrosequencing was run on a PyroMark Q24 (Qiagen, Valencia, CA). We used non-CpG cytosine residues as internal controls to verify efficient sodium bisulfite DNA conversion, and universal unmethylated (whole genome amplified) and methylated DNA (CpGenome Universal Methylated DNA, Millipore, Billerica, MA) were run as controls. Methylation quantification was performed using the PyroMark Q24 1.010 software. The degree of methylation was expressed for each DNA locus as percentage methylated cytosine over the sum of methylated and unmethylated cytosine. For LINE-1, values across the 3 CpG sites were averaged while for *IL-6*, values for the 6 sites were averaged. For LINE-1, the PCR primers and sequencing probe used were previously described (Bollati et al., 2007). For *IL-6*, the PCR primers and sequencing probe were designed to target sites within a CpG island located in the promoter region of the gene using the Pyromark Assay Design Software Version 2.0 (Qiagen). The sequences were as follows: TTTTGAGAAAGGAGGTGGGTAG (Forward PCR primer), ACCCCCTTAACCTCAAATCTACAATACTCT (5′ biotinylated Reverse PCR primer), and AAGGAGGTGGGTAGG (Sequencing primer). The coefficients of variation (CV) for the LINE-1 methylation assay range from 0.5 to 2.6% and the CVs for IL-6 promoter methylation assay range between 5.3 and 14.8%.

Dietary and physical activity measurements

We administered the validated 108-item Block food frequency questionnaire (FFQ), (Block et al., 1990; Subar et al., 2001) and the Block Adult Energy Expenditure Survey (Block et al., 2009). The nutrient and energy expenditure computations of the de-identified questionnaires were performed by NutritionQuest, the distributor of the two questionnaires.

Statistical analyses

We first compared the demographics between car drivers and PT users. Linear regression was used to estimate the difference and associated 95% confidence intervals (95%CI). We then compared the median and interquartile range (IQR) of daily intakes of foods and nutrients between the two groups. To construct dietary patterns, we performed factor analysis of 13 food groups using the principal factor method followed by an orthogonal rotation. Based on the scree test results, the proportion of variance accounted and the interpretability criteria, we identified two factors, i.e. two dietary patterns. For each subject, we estimated factor scores for the two dietary patterns by summing the frequency consumption of each food group weighted by their scoring coefficients. Subjects were then categorized into quartiles of factor scores for two dietary patterns, with high scores corresponding to a better adherence to a particular dietary pattern. We also estimated the car-vs-PT mean differences in factor scores for each of the two dietary patterns and associated 95%CIs using the beta coefficients of linear regression models and their standard errors. Next, we compared the median levels of reported daily physical activities between car drivers and PT users. Using linear regression, we also evaluated whether two groups differed in their adherence to physical activity guidelines by assessing the proportion of subjects meeting the U.S. Department of Agriculture 2005 Dietary Guidelines for Americans (DGA) for physical activity (i.e., engaged in approximately 60 min of moderate- to vigorous-intensity activity on most days of the week), or meeting the Healthy People 2010 Guidelines for physical activity (i.e., engaged in moderate physical activity for at least 30 min on at least 5 days a week, or engaged in vigorous physical activity for 20 min on at least 3 days/week). We used logistic regression to compare differences in distributions across quartiles of durations of the various types of physical activity.

Then, we compared the mean, median and adjusted geometric mean levels of CRP, WBC, LINE-1 methylation and *IL-6* promoter methylation between car drivers and PT users. Linear regression was used to estimate the difference and associated 95% confidence intervals. Because CRP levels did not follow a normal distribution, it was log-transformed in linear regression models.

Last, we created case–PT pairs of participants matched on age (±5 years) and gender and compared their differences in WBC, CRP, LINE-1 methylation and IL-6 methylation using paired-T tests. All statistical analyses were performed using SAS (version 9.1; SAS institute, Cary, NC).

Results

There were 79 car drivers and 101 PT users. Car drivers were older, had higher BMI, and included a greater proportion of males and non-Hispanic whites than PT commuters (Table 1).

Car drivers ate more fruits and more meats than PT users ($p = 0.02$ and 0.04 respectively, Table 2). We identified two dietary patterns in the study population: the prudent dietary pattern was characterized by high intakes of vegetables and fruits; and the western dietary pattern was characterized by high intakes of meats, grains and dairy products. Overall the two groups did not differ in the adherence to the two dietary patterns, either for the prudent or for the western diet (Table 3).

Car drivers reported a higher level of light job activities and a lower level of sedentary activities than PT commuters ($p = 0.007$ and 0.004 respectively). Overall, car drivers had higher adherence to 2005 DGA for physical activity than PT commuters (78.5% vs. 65.0%). However, after adjusting for age, gender, race/ethnicity and BMI, the difference in adherence to the 2005 DGA for physical activity

Table 1
Demographics of car drivers and public transportation (PT) users, New York, 2009–2010.

	Car	PT	Difference (95%CI)
N	79	101	
Age, *y*, mean (SD)	34.5 (15.6)	25.4 (8.9)	−9.2 (−12.8, −5.5)
Body Mass Index, *kg/m*^{2}, mean (SD)	26.0 (5.3)	24.2 (3.7)	−1.8 (−3.1, −0.5)
Women, n (%)	36 (45.6)	58 (57.4)	11.8 (−2.9, 26.6)
Race/ethnicity, n (%)			
Non-Hispanic whites	45 (59.2)	30 (32.6)	−26.6 (−41.4, −11.8)
Non-Hispanic blacks	9 (11.8)	16 (17.4)	5.5 (−5.4, 16.5)
Hispanics	15 (19.7)	29 (31.5)	11.8 (1.6, 25.2)
Others	7 (9.2)	17 (18.5)	9.3 (−1.4, 19.9)

CI = confidence interval.

Table 2
Food and nutrients differences by car versus public transportation (PT) commute modes. New York, 2009–2010.

	Car (n = 79)	PT (n = 101)
	Median (IQR)	
Food groups		
Vegetables (cups/day)	2.5 (1.3–4.8)	2.0 (1.1–3.5)
Leafy green vegetables (cups/day)	0.4 (0.2–0.7)	0.2 (0.1–0.6)
Orange/yellow vegetables (cups/day)	0.09 (0.05–0.2)	0.07 (0.04–0.1)
Legumes (cups/day)	0.1 (0.03–0.3)	0.2 (0.05–0.3)
Other vegetables (cups/day)	0.8 (0.5–1.3)	0.7 (0.4–1.1)
Starchy vegetables (cups/day)	0.2 (0.1–0.4)	0.2 (0.1–0.3)
Fruits (cups/day)	1.1 (0.8–0.4)	0.9 (0.5–1.6)
Total grains (oz./day)	5.0 (3.6–7.1)	4.8 (3.5–6.4)
Whole grains (oz./day)	1.1 (0.7–2.0)	1.0 (0.5–1.6)
Meats (oz./day)	2.9 (1.7–5.2)	2.2 (1.1–4.1)
Dairy (cups/day)	1.2 (0.7–1.6)	1.0 (0.6–1.7)
Oils (tsp./day)	2.6 (1.3–3.8)	2.4 (1.4–3.2)
Nutrients		
Total energy intake (kcal/day)	1836 (1302–2415)	1600 (1299–2135)
% Calorie from fat	35.0 (30.0–38.6)	33.3 (30.4–37.6)
% Calorie from saturated fat	10.0 (8.3–12.0)	9.7 (8.0–11.4)
% Calorie from *trans*–fat	0.9 (0.7–1.2)	1.0 (0.7–1.2)
Cholesterol (mg/day)	213.0 (131.7–307.8)	171.3 (120.5–292.9)
% Calorie from sweets	10.2 (5.8–16.9)	13.2 (7.0–20.3)
Glycemic index	50.6 (47.6–52.5)	51.0 (49.3–53.3)
Sodium (mg/day)	2802 (2072–3889)	2604 (1849–3533)
Fiber (g/1000 kcal)	9.6 (7.2–13.1)	9.2 (6.6–12.3)

IQR = interquartile range.

became statistically insignificant (difference: −14.2%, 95%CI: −29.0, 0.5) (Table 4).

In Table 5, there were no differences in median level of CRP (car vs. PT: 0.6 vs. 0.5 mg/dl, difference in log-CRP: 0.2, 95%CI: −0.2, 0.5) and mean level of WBC (car vs. PT: 6.7 vs. 6.5 cells/mm^3, difference: −0.4, 95%CI: −0.9, 0.2). In Table 6, there were no differences in mean levels of LINE-1 methylation (car: 78.0%; PT: 78.3%, difference: 0.2, 95%CI: −0.5, 1.0), and *IL-6* promoter methylation (car: 56.1%; PT: 58.0%, difference: 1.7, 95%CI: −2.4, 5.8). Missing values in Table 6 are due to low DNA yield following extraction from the buffy or low quality calls on pyrosequencing LINE-1 methylation or *IL-6* promoter methylation.

A total of 58 1-to-1, age-gender matched pairs comprising one PT commuter and one car commuter were formed. No statistically significant differences were found for WBC (difference = 0.07 cells/mm^3, 95%CI: −0.64, 0.77), CRP (difference = 0.03 mg/dl, 95%CI: −0.67, 0.74), LINE-1 methylation (difference = −0.07%, 95%CI: −0.91, 0.77) and IL-6 methylation (difference = −3.81%, 95%CI: −10.15, 2.52) between pairs. There remained, however, an age difference of about 1 year (difference = 0.98 year, 95% CI: 0.58, 1.39) within pairs.

Table 3
Differences in dietary patterns by car versus public transportation (PT) commute modes. New York, 2009–2010.

	Car (n = 79)	PT (n = 101)	
Dietary pattern factor scores (quartile range)			
Prudent dietary pattern			Difference (95% CI)[a]
Mean score (SD)	0.2 (1.2)	−0.1 (0.8)	−0.3 (−0.6, 0.05)
	N (%)		OR (95%CI)[a]
Q1 (<−0.7)	15 (19.0)	29 (29.0)	Ref.
Q2 (−0.7, −0.31)	20 (25.3)	25 (25.0)	1.3 (0.5, 3.4)
Q3 (−0.3, 0.49)	19 (24.1)	26 (26.0)	1.3 (0.5, 3.3)
Q4 (≥ 0.5)	25 (31.7)	20 (20.0)	2.1 (0.8, 5.8)
Western dietary pattern			
Mean score (SD)	0.03 (0.9)	−0.02 (0.9)	−0.03 (−0.3, 0.3)
	N (%)		OR (95%CI)[a]
Q1(<−0.6)	18 (22.8)	29 (26.0)	Ref.
Q2 (−0.6, −0.21)	18 (22.8)	27 (27.0)	0.8 (0.3, 2.1)
Q3 (−0.2, 0.39)	22 (27.9)	23 (23.0)	1.2 (0.5, 3.2)
Q4 (≥0.4)	21 (26.6)	24 (24.0)	1.3 (0.5, 3.4)

CI = Confidence Interval, OR = Odds Ratio, Q = Quartiles.
[a] Linear regression models were adjusted for age, gender, race/ethnicity and BMI.

Table 4
Difference in physical activity by car versus public transportation (PT) commute modes. New York, 2009–2010.

	Car	PT	
Physical activity levels (minutes/day)	Median (IQR)		
Moderate leisure activities	23.7 (5.4–50.7)	20.0 (2.7–52.1)	
	N (%)		OR (95%CI)[a]
Q1 (<3.9)	15 (19.0)	28 (28.0)	Ref.
Q2 (3.9, 23.7)	23 (29.1)	23 (23.0)	1.5 (0.5, 4.2)
Q3 (23.7, 51.6)	22 (27.9)	23 (23.0)	2.4 (0.9, 6.7)
Q4 (≥51.6)	19 (24.1)	26 (26.0)	2.1 (0.7, 6.2)
Moderate or vigorous leisure activities	46.8 (16.1–82.4)	33.7 (10.5–89.4)	
	N (%)		OR (95%CI)
Q1 (<12.3)	16 (20.3)	28 (28.0)	Ref.
Q2 (12.3, 36.7)	20 (25.3)	25 (25.0)	0.9 (0.3, 2.5)
Q3 (36.7, 85.5)	25 (31.7)	20 (20.0)	1.6 (0.6, 4.3)
Q4 (≥85.5)	18 (22.8)	27 (27.0)	1.3 (0.5, 3.5)
Moderate household activities	22.4 (5.4–59.0)	14.9 (5.4–29.2)	
	N (%)		OR (95%CI)
Q1 (<5.4)	19 (24.1)	24 (24.0)	Ref.
Q2 (5.4, 20)	14 (17.7)	32 (32.0)	0.5 (0.2, 1.2)
Q3 (20, 45)	22 (27.9)	22 (22.0)	1.2 (0.5, 3.0)
Q4 (≥45)	24 (30.4)	22 (22.0)	1.1 (0.4, 3.0)
Light job activities	300.0 (171.4–342.9)	205.7 (94.3–325.7)	
	N (%)		OR (95%CI)
Q1 (<128.6)	13 (16.5)	29 (28.0)	Ref.
Q2 (128.6, 214.3)	15 (19.0)	28 (28.0)	1.3 (0.5, 3.5)
Q3 (214.3, 342.9)	23 (29.1)	18 (18.0)	2.6 (1.0, 7.1)
Q4 (≥342.9)	28 (35.4)	25 (25.0)	1.5 (0.5, 3.9)
Sedentary activities[b]	241 (153–366)	311 (320–458)	
	N (%)		OR (95%CI)
Q1 (<225.9)	27 (34.2)	17 (17.0)	Ref.
Q2 (225.9, 325.8)	20 (25.3)	25 (25.0)	1.5 (0.5, 4.2)
Q3 (325.8, 476.1)	17 (21.5)	28 (28.0)	2.4 (0.9, 6.7)
Q4 (≥476.1)	15 (19.0)	30 (30.0)	2.1 (0.7, 6.2)
Adherence to physical activity guidelines	n (%)		*Differences (95% CI)*
Meet USDA 2005 DGA for physical activity	62 (78.5)	65 (65.0)	−14.2 (−29.0, 0.5)
Meet Healthy People 2010 Guidelines for physical activity	47 (59.5)	47 (47.0)	−11.2 (−27.8, 5.4)

CI = Confidence Interval, DGA = Dietary Guidelines for Americans, IQR = Interquartile Range (Q1–Q3), OR = Odds Ratio, Q = Quartiles.
USDA = US Department of Agriculture.
[a] Logistic regression and linear regression models were adjusted for age, gender, race/ethnicity and BMI.
[b] Minutes/day of sedentary activities include times spent watching TV or movie, on the internet or computer work, and sitting.

Discussion

In this first assessment of the biological impact of commute modes, we found that PT commuters to a New York college campus had a similar inflammatory and epigenetic status compared to car drivers, even though they were about 10 years younger and had 2 kg/m^2 lower BMI than the car commuters. However, compared to PT commuters, car drivers ate more fruits and were overall more physically active. These results are compatible with the American Time Use Survey (ATUS) which shows that daily commute tends to squeeze the time dedicated to other essential activities such as exercise, food preparation, and sleeping (Basner et al.,

Table 5
Differences in inflammatory response by car versus public transportation (PT) commute modes. New York, 2009–2010.

	Car	PT	*Differences*[a] *(95% CI)*
n	78	101	
CRP, mg/dl, mean (SD)	1.3 (1.9)	1.2 (1.7)	0.2 (−0.2, 0.5)
Median (IQR)	0.6 (0.2–1.7)	0.5 (0.2–1.5)	
Adjusted geometric means	0.7	0.7	
WBC, /mm^3, mean (SD)	6.7 (1.6)	6.5 (1.8)	−0.4 (−0.9, 0.2)
Median (IQR)	6.7 (5.6–7.4)	6.4 (5.4–7.4)	
Adjusted mean	6.7	6.5	

CI = Confidence Interval, IQR = Interquartite range.
[a] Linear regression adjusted for age, gender, race/ethnicity and BMI. Log transformation was performed for CRP in linear regression models.

2007; Christian, in press). A transportation survey conducted every year since 2007 in the study target population at Queens College has consistently shown that the median commute time of car drivers is 60 min, per day, versus 120 min for PT users (Morabia and Zheng, 2009). In a scenario in which car drivers commute in 1 h, and PT users in 2 h, ATUS predicts that the PT commuters will lack 2.2 min of exercise, 1.4 min of food preparation, and 15.6 min of sleeping per day (Christian, in press). The reduction of exercise time seems too modest to explain the present study results, but a compounded loss of 16.4 min per day in health-related activities (−5.2% for a two-hour commuter compared to a one-hour commuter) may make a difference. Thus, the time saved by car drivers in their commute can be allocated to health-related activities and may explain a higher adherence to physical activity guideline in car drivers than in PT commuters.

We explored differences in inflammatory response across commute modes because it is a plausible short-term effect of the type of moderate physical activity involved when commuting using PT. Physical activity can stimulate anti-inflammatory cytokine production, such as IL-1ra, IL-4 and IL-10, while sedentary behaviors can generate an excess of pro-inflammatory cytokines, such as IL-1, TNF and chemokines (Colbert et al., 2004). However, we did not find differences in CRP and WBC between two commute modes. Cytokine balance may be under epigenetic regulation (Backdahl et al., 2009). DNA methylation is an epigenetic event that may contribute to cancer and other human disease occurrence by altering gene expression. Global hypomethylation, as indicated by low levels of LINE-1 methylation, has been associated with genome instability and elevated cancer risk, whereas methylation in the promoter region of specific genes is associated with gene silencing. Methylation patterns can be influenced by environmental factors such as diet, (Zhang et al., 2011b) physical activity, (Bjornsson et al., 2008; Coyle et al., 2007; Zhang et al., 2011a) and air pollution (Miller and Ho, 2008). In this study, we did not find that commuting modes affected the methylation levels of LINE-1 or *IL-6* promoter. The lack of difference in global CRP, WBC, and methylation levels between car drivers and PT users may be due to other characteristics associated with their commute or their way of life. For example, our previous work indicates a slight increase in exposure to $PM_{2.5}$ for a 7 h trip by PT (mostly subway) vs. by car, (Morabia et al., 2009) and air pollution increases inflammatory response (Pope et al., 2004). Short-term (Liao et al., 2005; Schwartz, 2001) and long-term (Chen and Schwartz, 2008) elevation of ambient PM_{10} is associated with increased levels of inflammatory markers (Peters et al., 2001; Pope et al., 2004).

Table 6
Differences in DNA methylation by car versus public transportation (PT) commute modes. New York, 2009–2010.

	Car	PT	
LINE-1 methylation, n	72	93	
			Difference[a] (95% CI)
%, Mean (SD)	78.0 (2.1)	78.3 (2.0)	0.2 (−0.5, 1.0)
Median (IQR)	78.4 (77.2–79.6)	78.3 (77.3–79.3)	
Adjusted means	78.1	78.2	
Quartiles (%)	n (%)	n (%)	OR (95%CI)
Q1 (<77.2)	18 (25.0)	21 (22.6)	Ref.
Q2 (77.2, 78.2)	17 (23.6)	25 (26.9)	0.6 (0.2, 1.7)
Q3 (78.3, 79.4)	15 (20.8)	25 (26.9)	0.6 (0.2, 1.6)
Q4 (≥79.5)	22 (30.6)	22 (23.7)	1.1 (0.4, 2.8)
IL-6 promoter methylation, N	72	85	*Difference*[a] (95% CI)
%, Mean (SD)	56.1 (11.8)	58.0 (12.1)	1.7 (−2.4, 5.8)
Median (IQR)	56.0 (47.6–63.5)	55.6 (50.9–66.1)	
Adjusted means	55.6	58.4	
Quartiles (%)	n (%)	n (%)	OR (95%CI)
Q1 (<49.8)	21 (29.2)	18 (21.2)	Ref.
Q2 (49.8, 55.8)	14 (19.4)	25 (29.4)	0.4 (0.1, 1.0)
Q3 (55.9, 64.5)	20 (27.8)	19 (22.4)	0.7 (0.2, 1.8)
Q4 (≥64.6)	17 (23.6)	23 (27.1)	0.5 (0.2, 1.3)

CI = Confidence Interval, IQR = Interquartile Range, OR = Odds Ratio.
[a] Linear regression and logistic regression models were adjusted for age, gender, race/ethnicity and BMI.

Limitations

As our previous research has already shown that PT commuters to Queens College expend more energy than car commuters, the physical activity questionnaire for the current study was mainly designed to assess the physical activity of the participants beyond their commute. We therefore did not have the possibility to factor out the specific extra energy spent during the commute in these analyses. Our results, however, indicate that future studies should use a more detailed measure of physical activity, such as diaries, in order to decompose it into commute, leisure, home, and work.

Limitations in the methodologies used to determine biomarker levels may have also hampered our ability to identify an association with commute mode. For the assessment of *IL6* gene promoter methylation, the variability across the sites targeted within the *IL6* promoter, as indicated by the coefficient of variation, may have reduced the robustness of the designed assay to capture the acute differences to be expected within this setting. Similarly, assay-based issues may have impacted the assessment of global methylation. LINE-1 is a retrotransposon distributed throughout the genome. As a repetitive element, it can be easily assessed using a PCR-based method, making it amenable for population-based studies. However, though commonly used, it has not been established how adequately this surrogate marker reflects true genome-wide methylation levels.

A strength of this study was its sampling method since participants were randomly selected, according to their commute type and duration, from a roster of about 4000 persons who previously provided a detailed description of their commute mode in repeated college-wide surveys. Its design, analogous to a case–control study in which car drivers are the "cases" and PT commuters the "controls," provides insight into potential differential selection processes. In particular, PT commuters responded better than car drivers to each of the multiple emails sent to all the eligible subjects. Our objective of 100 PT users was easily met, but we were not able to recruit during the same period more than 79 car drivers. We cannot therefore rule out that car drivers were selected among a more physically active and health conscious subset of the target population, therefore attenuating the observed differences.

These results need to be considered in a context of growing interest in public transportation as a means of reducing fossil-fuel consumption and global warming (Zheng, 2008). Americans took, in 2007, 10.3 billion PT trips, representing a 32% increase compared to 1995. Between January and September 2008, PT usership increased, for example, by 3.8% in New York, 8.1% in Atlanta, and 32.7% in Charlotte, NC (APTA, 2008). Plans of developing a rapid rail network across the US are under discussion.

The similar inflammatory and epigenetic traits observed in this study in car and PT commuters convey an important and apparently neglected

prevention message that, if not integrated into a more general strategy to achieve overall dietary and physical activity objectives, society may miss the health benefit to be harvested if commute modes increasingly are switched from car to PT.

Conflict of interest statement

None of the authors have conflict of interests with the content of the paper.

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References

APTA, 2008. Public transportation fact book. http://www.apta.com/research/stats/factbook/documents08/2008_fact_book_final_part_1.pdf. 2008. American Public Transportation Association.

Backdahl, L., Bushell, A., Beck, S., 2009. Inflammatory signalling as mediator of epigenetic modulation in tissue-specific chronic inflammation. Int. J. Biochem. Cell Biol. 41, 176–184.

Basner, M., Fomberstein, K.M., Razavi, F.M., et al., 2007. American time use survey: sleep time and its relationship to waking activities. Sleep 30, 1085–1095.

Besser, L.M., Dannenberg, A.L., 2005. Walking to public transit: steps to help meet physical activity recommendations. Am. J. Prev. Med. 29, 273–280.

Bjornsson, H.T., Sigurdsson, M.I., Fallin, M.D., et al., 2008. Intra-individual change over time in DNA methylation with familial clustering. JAMA 299, 2877–2883.

Block, G., Woods, M., Potosky, A., Clifford, C., 1990. Validation of a self-administered diet history questionnaire using multiple diet records. J. Clin. Epidemiol. 43, 1327–1335.

Block, G., Jensen, C.D., Block, T.J., et al., 2009. The work and home activities questionnaire: energy expenditure estimates and association with percent body fat. J. Phys. Act. Health 6, S61–S69.

Bollati, V., Baccarelli, A., Hou, L., et al., 2007. Changes in DNA methylation patterns in subjects exposed to low-dose benzene. Cancer Res. 67, 876–880.

Chen, J.C., Schwartz, J., 2008. Metabolic syndrome and inflammatory responses to long-term particulate air pollutants. Environ. Health Perspect. 116, 612–617.

Christian, T., in press. Trade-offs between commuting time and health-related activities. J. Urban Health.

Colbert, L.H., Visser, M., Simonsick, E.M., et al., 2004. Physical activity, exercise, and inflammatory markers in older adults: findings from the Health, Aging and Body Composition Study. J. Am. Geriatr. Soc. 52, 1098–1104.

Coussens, L.M., Werb, Z., 2002. Inflammation and cancer. Nature 420, 860–867.

Coyle, Y.M., Xie, X.J., Lewis, C.M., et al., 2007. Role of physical activity in modulating breast cancer risk as defined by APC and RASSF1A promoter hypermethylation in nonmalignant breast tissue. Cancer Epidemiol. Biomarkers Prev. 16, 192–196.

Edwards, R.D., 2008. Public transit, obesity, and medical costs: assessing the magnitudes. Prev. Med. 46, 14–21.

Ford, E.S., 2002. Does exercise reduce inflammation? Physical activity and C-reactive protein among U.S. adults. Epidemiology 13, 561–568.

Hammett, C.J., Oxenham, H.C., Baldi, J.C., et al., 2004. Effect of six months' exercise training on C-reactive protein levels in healthy elderly subjects. J. Am. Coll. Cardiol. 44, 2411–2413.

Hill, J.O., Wyatt, H.R., Reed, G.W., Peters, J.C., 2003. Obesity and the environment: where do we go from here? Science 299, 853–855.

Kasapis, C., Thompson, P.D., 2005. The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review. J. Am. Coll. Cardiol. 45, 1563–1569.

Lachapelle,, u., Frank, L.D., 2009. Transit and health: Mode of transport, employer-sponsored public transit pass programs, and physical activity. J. Public Health Policy 30, S73–S94.

Liao, D., Heiss, G., Chinchilli, V.M., et al., 2005. Association of criteria pollutants with plasma hemostatic/inflammatory markers: a population-based study. J. Expo. Anal. Environ. Epidemiol. 15, 319–328.

MacDonald, J.M., Stokes, R.J., Cohen, D.A., Kofner, A., Ridgeway, G.K., 2010. The effect of light rail transit on body mass index and physical activity. Am. J. Prev. Med. 39, 105–112.

Miller, R.L., Ho, S.M., 2008. Environmental epigenetics and asthma: current concepts and call for studies. Am. J. Respir. Crit. Care Med. 177, 567–573.

Mora, S., Cook, N., Buring, J.E., Ridker, P.M., Lee, I.M., 2007. Physical activity and reduced risk of cardiovascular events: potential mediating mechanisms. Circulation 116, 2110–2118.

Morabia, A., Costanza, M.C., 2004. Does walking 15 minutes per day keep the obesity epidemic away? Simulation of the efficacy of a populationwide campaign. Am J. Public Health 94, 437–440.

Morabia, A., Zheng, Y., 2009. On the influence of a raffle upon responses to an urban transportation survey in New York City. Int J. Public Health 54, 31–34.

Morabia, A., Amstislavski, P.N., Mirer, F.E., et al., 2009. Air pollution and activity during transportation by car, subway, and walking. Am. J. Prev. Med. 37, 72–77.

Morabia, A., Mirer, F.E., Amstislavski, T.M., et al., 2010. Potential health impact of switching from car to public transportation when commuting to work. Am. J. Public Health 100, 2388–2391.

Ogilvie, D., Egan, M., Hamilton, V., Petticrew, M., 2004. Promoting walking and cycling as an alternative to using cars: systematic review. BMJ 329, 763.

Peters, A., Frohlich, M., Doring, A., et al., 2001. Particulate air pollution is associated with an acute phase response in men; results from the MONICA-Augsburg Study. Eur. Heart J. 22, 1198–1204.

Pope III, C.A., Hansen, M.L., Long, R.W., et al., 2004. Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. Environ. Health Perspect. 112, 339–345.

Rogers, C.J., Colbert, L.H., Greiner, J.W., Perkins, S.N., Hursting, S.D., 2008. Physical activity and cancer prevention: pathways and targets for intervention. Sports Med. 38, 271–296.

Schwartz, J., 2001. Air pollution and blood markers of cardiovascular risk. Environ. Health Perspect. 109, 405–409.

Subar, A.F., Thompson, F.E., Kipnis, V., et al., 2001. Comparative validation of the Block, Willett, and National Cancer Institute food frequency questionnaires: the Eating at America's Table Study. Am. J. Epidemiol. 154, 1089–1099.

Zhang, F.F., Cardarelli, R., Carroll, J., et al., 2011a. Physical activity and global genomic DNA methylation in a cancer-free population. Epigenetics 6, 293–299.

Zhang, F.F., Morabia, A., Carroll, J., et al., 2011b. Dietary patterns are associated with levels of global genomic DNA methylation in a cancer-free population. J. Nutr. 141, 1165–1171.

Zheng, Y., 2008. The benefit of public transportation: physical activity to reduce obesity and ecological footprint. Prev. Med. 46, 4–5.